the amount of trapped cells and their viability. There were no acute complications (death, tamponade, ventricular perforation, arrhythmia). Preliminary in vitro testing of cell viability after passage through the catheter yielded a 88.6 +/- 2.5% (mean +/- SEM) rate which was highly pressure-dependent. Following explantation, clusters of SM could be histologically identified by computerized assisted analysis in the anterior, apical and septal walls in the 6 hearts and represented 11 +/- 2.2% of the initial injectate. The viability of grafted SM retrieved from the transplanted area and assessed by a positive staining for CD 56 averaged 3%. This percentage which might represent an underestimation due to injection in non infracted myocardium, is still 10 fold higher than that previously observed after SM injections through an endovascular catheter. These preliminary data suggest that the CS route could represent a safe and effective mean of percutaneous SM intramyocardial transfer but improvements in delivery systems remain mandatory to reduce physical train during injections and consequently optimise cell survival.

1176-197 Combination of a Direct Thrombin Inhibitor, Argatroban, and Glycoprotein IIb/IIIa Inhibitor Is Effective and Safe in Patients Undergoing Percutaneous Coronary Intervention

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Background: Argatroban, a small molecule direct thrombin inhibitor, has been shown to block clot bound thrombin more effectively than does unfractionated heparin. Argatroban has not been systematically tested in patients undergoing percutaneous coronary intervention (PCI) with concurrent glycoprotein (GP) IIb/IIIa.
Methods: In this multicenter, prospective, pilot study argatroban was administered in patients undergoing PCI at 250 mcg/kg bolus followed by 15 mcg/kg/min during the procedure. Additional boluses of 150 mcg/kg were given, if ACT did not reach the target range of 275-325 sec. GP IIb/IIIa inhibitor was administered simultaneously. The primary efficacy endpoint was vascular death, myocardial infarction (MI), or urgent revascularization at 30 days. The safety endpoint was in-hospital major bleeding. MI was defined as CKMB elevation more than 3 times the upper limit of normal or cardiac symptoms with supportive cardiac marker or EKG evidence.
Results: A total of 101 patients were enrolled and completed the PCI procedure. There were 72 males and the mean age was 65 years. Abciximab was given to 99 patients and double bolus eptifibatide to 2 patients. 76 patients had one target lesion treated and the rest, two or more lesions. 96 patients were treated with stenting. Second and third boluses of argatroban were required in 22 and 7 patients, respectively. The target ACT was achieved in 94 patients. The primary efficacy endpoint occurred in 3 (3.0%) patients (no vascular death, 3 MIs and 2 urgent revascularizations). Two additional patients had cardiac symptoms and elevated troponin without significant CKMB elevation. There were 2 major bleeding events (1 retroperitoneal, 1 groin hematoma). **Conclusion:**Argatroban in combination with GP IIb/IIIa inhibitors provides adequate anticoagulation with acceptable bleeding risk. These data suggest that further investigation of argatroban in patients undergoing PCI is warranted.

1176-198 Unfractionated Heparin Increases Platelet-Monocyte Binding In Vitro and in Patients Undergoing Percutaneous Coronary Intervention

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Background: Unfractionated heparin is currently considered to be the anticoagulant of choice in patients undergoing percutaneous coronary intervention (PCI). Unfractionated heparin may be associated with adverse platelet activation. Platelet-monocyte aggregation (PMA) is a sensitive marker of platelet activation and may mediate pro-inflammatory cytokine release, tissue factor expression and augmented adhesion molecule expression. We compared the effects of unfractionated heparin, a low-molecular-weight-heparin (enoxaparin), and a direct thrombin inhibitor (lepirudin) on PMA in vitro. We also investigated the effects of unfractionated heparin on PMA in patients prior to PCI.
Methods: Peripheral venous blood was collected from 18 healthy volunteers into sodium citrate alone or sodium citrate with unfractionated heparin (1 U/ml), enoxaparin (0.8 U/ml) or lepirudin (5.6 μg/ml). Blood was also drawn into sodium citrate tubes from 14 patients immediately before and five minutes after administration of 100 U/kg of unfractionated heparin, prior to elective PCI. PMA was determined by 2-color flow-cytometric analysis.
Results: In healthy volunteers, PMA was higher in blood anticoagulated with sodium citrate and unfractionated heparin (20.1 ± 1.9%) compared to sodium citrate alone (16.2 ± 1.6%, p<0.001) or sodium citrate with enoxaparin (16.9 ± 2.0%, p<0.01) or lepirudin (17.0 ± 2.2%, p<0.01). There were no differences in PMA in blood anticoagulated with sodium citrate alone, sodium citrate and enoxaparin, or sodium citrate and lepirudin (p=ns). Administration of unfractionated heparin to patients prior to PCI was also associated with increased PMA (24.2 ± 2.6% vs. 16.9 ± 2.4%, p<0.01).
Conclusions: In contrast to enoxaparin and lepirudin, unfractionated heparin increases PMA in vitro; an effect that was also demonstrable in patients receiving unfractionated heparin prior to PCI. Given that PMA is a sensitive measure of platelet activation and may have proinflammatory consequences, the use of alternative anticoagulant regimens may reduce PCI associated complications.

1176-199 Safety and Efficacy of Subcutaneous Enoxaparin in Early Invasive Strategy of Unstable Angina

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Introduction : We have demonstrated previously that subcutaneous (s/c) enoxaparin (1mg/Kg/12h) given during at least 48 hours provided good anticoagulation and clinical results in non-ST elevation acute coronary syndromes (NSTE-ACS) patients undergoing percutaneous coronary intervention (PCI) within 8 hours of the last injection. We evaluated whether an early invasive (EI) strategy with only 2 injections of s/c enoxaparin was as good as a delayed invasive (DI) strategy with 3 injections or more.
Methods and results : We compared NSTE-ACS patients who underwent PCI after 2 injections of s/c enoxaparin (EI, n=117) with those referred later on (DI, 5.9±0.2 s/c injections, n=230). Anti-Xa at the time of catheterization, safety and major coronary events (death/MI) were assessed at 30 days. Baseline characteristics were similar in the 2 groups of patients. The period of medical stabilization was 20.5±1.0 hrs in the "EI" and 69.2±3.0 hrs in the "DI" group, respectively (p<0.0001). The anti-Xa activity measured at the time of catheterization (0.92±0.04 U/mL vs 0.96±0.02 U/mL, p=0.25) and the injection to catheterization time (5.6±0.2 hrs. vs 5.2±0.1 hrs, p=0.17) were similar in both groups. Patients of the "EI" group were more frequently treated by IIb/IIIa inhibitors and clopidogrel before PCI than patients of the "DI" group (58.1% vs 31.7%, p<0.0001 for GPIIb/IIIa inhibitors and 68.4% vs 40.4% for clopidogrel pretreatment, p<0.0001, respectively). Bleeding rates were found to be equivalent between both groups (1.7% vs 4.8%, for "EI" and "DI" strategies respectively, p=0.23, one-sided 95% CI of 0.826). There was a non significant trend for less death or MI at 30 days in the "EI" group compared to the "DI" group (4.3% vs 7.0%, respectively, p=0.32, one-sided 95% CI of 1.536).
Conclusion: In patients with NSTE-ACS, a rapid invasive strategy with only 2 s/c injections of enoxaparin provides similar levels of anticoagulation and similar bleeding rates with a trend for less ischemic major events as a more prolonged "upstream" treatment with enoxaparin.

1176-200 Assessment of Anticoagulation Using Activated Clotting Times in Patients Receiving Intravenous Enoxaparin During Percutaneous Coronary Intervention

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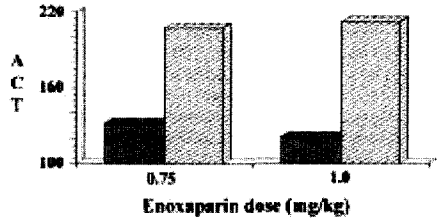
Background: Low molecular weight heparins have many advantages over unfractionated heparin. They have been used safely during percutaneous coronary intervention (PCI) when given intravenously (IV) with and without glycoprotein 2b/3a receptor inhibitors. Interventionalists have been reluctant to use this therapy because of the inability to monitor the anticoagulant effect of IV enoxaparin. We measured activated clotting time (ACT), before and after IV enoxaparin, to determine if anticoagulation could be assessed with this standard test.
Methods: 45 consecutive patients undergoing PCI received either 0.75 mg/kg IV enoxaparin if they also received eptifibatide, or 1 mg/kg IV enoxaparin if no eptifibatide was given. ACT was measured using the Hemochron device before and 5 minutes following IV enoxaparin administration.
Results: After 0.75 mg/kg enoxaparin, mean ACT increased from 132 ± 31 sec to 207 ± 25 sec, (p<0.001). After 1 mg/kg enoxaparin, ACT increased from 121 ± 28 sec to 212 ± 32 sec, (p<0.001). The mean increase in ACT value was 74 ± 20 sec (range 47 to 132) in the 0.75 mg/kg group (n=36) and 92 ± 28 sec (range 32 to 120) in the 1 mg/kg group (n=9). None of the patients had transient abrupt closure, thrombus formation, major bleeding or required urgent revascularization.



Conclusions: Intravenous enoxaparin at clinically relevant doses, increases ACT levels in patients undergoing PCI with and without eptifibatide. These data suggest that ACT may be useful in the measurement of enoxaparin anticoagulation.

1176-201 A Randomized Comparison of Dalteparin Versus Unfractionated Heparin During Percutaneous Coronary Interventions

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Background: Low molecular weight heparins are widely used in patients with coronary artery disease but experience with them during percutaneous coronary interventions (PCI) is limited. The purpose of this study was to compare the safety and efficacy of the low molecular weight heparin, dalteparin (Dalt.) to unfractionated heparin (UFH).
Methods: A single-centre, randomized, double-blind study of Dalt. versus UFH in patients undergoing PCI was carried out. All patients undergoing planned or ad-hoc PCI (excluding emergency PCI post-thrombolysis or for shock) were eligible. Randomization was